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The relationship of exercise and diet to total cholesterol and high density lipoprotein-cholesterol college age males and females

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THE RELATIONSHIP OF EXERCISE AND DIET TO TOTAL CHOLESTEROL AND HIGH DENSITY LIPOPROTEIN-CHOLESTEROL IN COLLEGE AGE MALES AND FEMALES

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by

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CHAPTER I

Introduction

Atherosclerosis is one of the biggest concerns in the medical and health profession today. Thirty-three percent of all deaths in this country can be attributed to degenerative and arteriosclerotic heart disease (Berkow, 1982). Cerebral vascular disease, which has been linked to atherosclerosis, is the third major cause of death in the United States, behind only heart disease and cancer (Berkow, 1982).

Atherosclerosis is a disease of the arteries and is defined as a form of arteriosclerosis in which fatty lesions called atheromatous plaques form on the intima of arteries. The formation of these plaques begins early, within the first two decades of life, and may be started by damage to the endothelial cells and intima of the artery walls (Guyton, 1981). A number of factors may cause the initial damage, including physical abrasion of the endothelium, abnormal substances in the blood or pulsating arterial pressure on the vessel wall (Guyton, 1981).

A number of important factors have been identified as possible contributors to the development

of atherosclerosis, including: hypertension, elevated serum lipids, cigarette smoking, diabetes mellitus, obesity, physical inactivity, hereditary disorders, age and gender (Guyton, 1981). A principal concern to the physical educator is the role of exercise and diet in the prevention and treatment of atherosclerosis.

There is a growing body of epidemiologic, genetic, experimental and clinical evidence to support the hypothesis that there is a cause and effect relationship between high blood levels of cholesterol and the development of atherosclerosis in humans.

Statement of the Problem

The purpose of this study was to determine the relationship of exercise and diet in predicting the total cholesterol/high density lipoprotein-cholesterol (TC/HDL-C) ratio in college age males and females. Variables controlled for included age, gender, smoking, medication use, contraceptive use, hormone use and intense physical activity.

Variables considered in the equation included exercise, the percentage of total fat in the diet, the ratio of polyunsaturated to saturated fats in the diet, and the amount of cholesterol in the diet.

The components selected as subproblems were the following:

1. What is the relative importance of frequency, duration and intensity of exercise (after controlling for all other factors) in predicting the TC/HDL-C ratio in college age males and females?

2. What is the relative importance of the percentage of total fat in the diet (after controlling for all other variables) in predicting the TC/HDL-C ratio in college males and females?

3. What is the relative importance of the ratio of polyunsaturated to saturated fats in the diet (after controlling for all other variables) in predicting the TC/HDL-C ratio in college age males and females?

4. What is the relative importance of the amount of cholesterol in the diet (after controlling for all other variables) in predicting the TC/HDL-C ratio in college age males and females?

5. Are any of the interactions between these exercise and dietary variables and their ability to predict the TC/HDL-C ratio different for males than for females in this college age population?

Importance of the Study

A number of studies have shown that high TC/HDL-C ratios in children and young adults translate to high TC/HDL-C ratios in later life and to the subsequent increased risk to develop cardiovascular disease (Freedman et al., 1985; Green et al., 1985; Mellies et al., 1985; Newman et al., 1986; Orchard et al., 1985). The most recent studies in this area have concentrated on elucidating the factors that influence the TC/HDL-C ratio.

Both chronic and acute exercise have been shown to affect the blood lipid and cholesterol levels. Much of the research in this area, however, has been limited in sample size or confounded by uncontrolled factors; this may or may not interact with exercise to produce the obtained results.

Studies examining the effects of diet on blood lipids have consistently found a positive relationship between the amount of fat, particularly saturated fat, in the diet and increased risk of coronary heart disease (Brunner and Lobl, 1958; Connor, 1961; DeBakey, 1984; Ernst et al., 1980; Fejfar and Masironi, 1970; Glueck and Connor, 1978; Kagan et al, 1974).

These studies, however, have concentrated mainly on older males, leaving out large portions of the entire population.

In the pilot for this study (Rothschild, 1985), it was found that three out of the ten subjects tested had cholesterol levels that would be considered moderate to high risk for their age group. This, in addition to the above mentioned inadequacies of previous studies, illustrates the importance of studying a younger population to determine the relative importance and various interrelationships among the exercise and dietary variables.

We may learn how to better protect ourselves from this killer disease by understanding more clearly the predictive abilities of exercise and dietary factors in a younger population in which the future development of coronary heart disease is just beginning to show.

Delimitations of the Study

The study was delimited in the following manner:

1. All subjects were between 18 and 23 years of age.

2. All subjects using hormones, contraceptives

or any lipid-affecting medication were excluded from the study.

3. All subjects were non-smokers.

4. All subjects participating in intense physical activity just prior to testing (within three days) were excluded from the study.

5. All subjects were University of the Pacific students.

Limitations of the Study

This study was limited in the following respects:

1. The researcher recognized the difficulty of obtaining precise and accurate information from a self-report method.

2. The researcher did not control for genetic factors and individual differences.

Hypotheses

The following hypotheses were investigated:

1. There would not be a linear relationship between exercise and diet and the TC/HDL-C ratio.

2. There would not be a linear relationship between frequency, duration and intensity of exercise (after controlling for all other factors) and the TC/HDL-C ratio.

3. There would not be a linear relationship

between the percentage of total fat in the diet (after controlling for all other factors) and the TC/HDL-C ratio.

4. There would not be a linear relationship between the ratio of polyunsaturated to saturated fats in the diet (after controlling for all other variables) and the TC/HDL-C ratio.

5. There would not be a linear relationship between the amount of cholesterol in the diet (after controlling for all other variables) and the TC/HDL-C ratio.

6. There would not be a significant difference between the ability of any variable or combination of variables to predict the TC/HDL-C ratio for males when compared to females in this college age population.

Basic Assumptions

The study was based on these assumptions:

1. All subjects recorded diet and exercise patterns accurately and honestly.

2. All subjects answered control questions accurately and honestly.

3. Genetic factors and individual differences would not significantly affect this study.

Definition of Terms

For the purpose of this study, the following terms were defined:

Concentrations in Human Plasma. Concentrations in human plasma refers to milligrams of cholesterol or HDL-C in 100 mililiters of plasma.

Intense Acute Exercise. Intense acute exercise refers to exercise well above the individual's established exercise patterns.

Abbreviations

The following abbreviations are found in the text:

1. CHD is coronary heart disease.
2. CHOL represents dietary cholesterol.
3. FAT represents the percentage of fat in the diet.
4. HDL-C is high density lipoprotein-cholesterol.
5. Kcal/kg/min refers to kilocalories per kilogram per minute.
6. LDL-C is low density lipoprotein-cholesterol.
7. LRC-CPPT is the Lipid Research Clinics Coronary Primary Prevention Trial.
8. LRCP is the Lipid Research Clinics Program.

9. Mg/dl refers to milligrams per deciliter.

10. mmHg represents millimeters of Mercury.

11. MRFIT is the Multiple Risk Factor Intervention Trial.

12. NIH represents the National Institutes of Health.

13. PA represents physical activity.

14. PPRG represents the Pooling Project of the Framingham Study Research Group.

15. RATIO represents the total cholesterol/high density lipoprotein-cholesterol ratio.

16. SAT represents the saturated/unsaturated fat ratio in the diet.

17. TC/HDL-C is the total cholesterol/high density lipoprotein-cholesterol ratio.

18. WDTB refers to the Worthington Diagnostics Technical Bulletin.

19. WHO is the World Health Organization.

CHAPTER II

Review of the Related Literature

Review of the literature uncovered a number of studies linking diet and exercise to the increased risk of developing atherosclerosis in older populations (Brunner and Lobl, 1958; Connor, 1961; Dayton et al., 1969; Fejfar and Masironi, 1970; Glueck and Connor, 1978; Gofman et al., 1966; Hartley, 1985). Many recent studies (Mellies et al., 1985; NIH, 1985; Newman et al., 1986) demonstrate a relationship between blood cholesterol levels and early atherosclerotic development in younger populations.

Review of the related literature includes the following: (1) the relationship between total blood cholesterol and coronary heart disease, (2) the importance of high density lipoprotein-cholesterol, (3) risk factors associated with elevated cholesterol and coronary heart disease, (4) the role of exercise in decreasing coronary heart disease risk and (5) tracking studies: the importance of identifying risk patterns at an early age.

The Relationship Between Total Blood Cholesterol and Coronary Heart Disease

There are four major types of research that have been conducted on the relationship between blood levels of cholesterol and atherosclerotic development. These are (1) clinical intervention, (2) epidemiological, (3) genetic and (4) experimental research. All strongly support a causal relationship between elevated cholesterol and the development of atherosclerosis.

The Oslo Heart Study (Hjermann, 1982) was a clinical intervention study that intervened on two risk factors associated with coronary heart disease; hypercholesterolemia and smoking. Intervention cases successfully altered both risk factors and, when compared to controls, showed a concomittant reduction in the incidence of coronary heart disease.

The World Health Organization (WHO) European Collaborative Group's Multifactorial Trial in the Prevention of Coronary Heart Disease (WHO, 1983), studied the effects of intervention on several risk factors in the hopes of reducing fatal coronary heart disease. The intervention program included counseling on dietary modification, cessation of cigarette smoking, weight

reduction, daily exercise, and treatment of hypertension (Borhani, 1985).

After seven years, the incidence of fatal coronary heart disease was reduced with intervention by 7.4%, a non-significant reduction. Problems with design and control of the WHO study have been cited as the major reasons for the lack of any conclusive results (Borhani, 1985).

The Multiple Risk Factor Intervention Trial (MRFIT) tested the hypothesis that special intervention in hypercholesterolemia, hypertension, and smoking among high-risk men would result in a significant reduction in mortality from coronary heart disease as compared with usual intervention by practicing physicians (MRFIT, 1982).

After a six-year trial period, results showed a non-significant 7.1% reduction in coronary heart disease mortality among the special intervention group, compared with the usual care group. Careful analysis of the MRFIT study suggests that the reason for the non-significance of the results could have been that there was no difference between the two groups in physician's care (Borhani, 1985).

The Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT) was a ten-year, double-

blind clinical intervention trial that tested the hypothesis that reduction in blood cholesterol alone would significantly reduce mortality from coronary heart disease (LRC-CPPT, 1984). This study recruited 3,806 middle-aged asymptomatic men with an average cholesterol level of 265 mg/dl or above.

All participants were prescribed diets designed to lower blood cholesterol levels three to four percent. Participants were later randomly divided into two treatment groups with the first group given a bile acid sequestrant cholestyramine resin and the second group given an equivalent amount of placebo.

Both groups continued to receive dietary counseling prescribed prior to randomization and all participants were followed up for seven to ten years. The primary end point of the LRC-CPPT study was the combination of definite fatal or definite non-fatal myocardial infarction.

The combination of diet and cholestryamine was successful in lowering the average plasma cholesterol level by 8.5 percentage points more than the diet and placebo, and the incidence of coronary heart disease was 19% lower in the cholestryamine treated group (p. less than .05).

In the recent National Institutes of Health Concensus Development Conference to Lower Blood Cholesterol to Prevent Heart Disease, the issue of extrapolation from the LRC-CPPT results was extensively debated (NIH, 1985). The pannel concluded that the results of the LRC-CPPT demonstrated conclusively that the lowering of blood cholesterol will reduce the incidence of coronary heart disease and that these benefits could be extrapolated to other groups.

Others have disagreed, however, saying that the trial did not prove that lowering cholesterol levels saves lives and that there is no basis for extending these results to those with lower blood cholesterol levels (Kolata, 1985; Kronmal, 1985).

In reply, the Lipid Research Clinics Program investigators pointed out that an overwhelming amount of corroborating evidence for genetic, experimental pathology, epidemiologic and clinical trial investigations supports the etiologic role of cholesterol in atherosclerotic coronary heart disease (LRCP, 1985). Furthermore, investigators have found that cholesterol's role in increasing CHD incidence exists even at lower blood concentrations than examined in the LRC-CPPT study (LRCP, 1985).

One recent study (Neaton et al., 1984) found that the risk of coronary heart disease mortality is a function of plasma cholesterol levels from 180 mg/dl upward, as had been suggested earlier from the results of clinical research on animals (Guyton, 1981).

The LRC-CPPT study was further examined for both internal and external consistency (LRCP, 1985). In testing for internal consistency, an average 19% reduction in incidence of CHD as a result of a 22.3 mg/dl (10.4%) decrement in cholesterol levels was compared to a predicted 16% reduction in incidence of CHD as a result of a 22.3 mg/dl fall in cholesterol levels.

For external consistency, the LRC-CPPT data was compared to the findings of both the Pooling Project of the Framingham Study (PPRG, 1978) and the Western Collaborative Study (Keys et al., 1972). These studies predicted an 11 to 19% reduction in coronary heart disease incidence for a 20.7 mg/dl decrement in cholesterol levels. The LRC-CPPT findings showed a 15.1% drop in CHD incidence (LRCP, 1984).

A number of other studies were further compared to the LRC-CPPT study in the form of a regression equation including the findings from

all studies (LRCP, 1984). The regression lines projected a 15.3% reduction (for diet) and a 20.9% reduction (for drug) in CHD incidence per ten percent decrement in total cholesterol levels. These predictions showed a strong relationship to the observed findings in the LRC-CPPT study (LRCP, 1981).

Several important epidemiologic studies, including the Framingham Study (Castelli et al., 1977) and the Western Collaborative Study (Keys, 1970) have supported the relationship between cholesterol and incidence of coronary heart disease as indicated in the clinical intervention trials. The Framingham Study was a case-control study involving 6,859 men and women of black, Japanese and white ancestry aged 40 years and older.

These subjects were chosen from populations in Albany, Framingham, Evans County, Honolulu and San Francisco. Cholesterol levels were significantly elevated in individuals who showed signs of CHD and there was a significant correlation between blood choelsterol and coronary heart disease prevalence (Castelli et al., 1977).

The Western Collaborative Study compared blood cholesterol levels and incidence of coronary heart disease in seven different countries

and showed a strong correlation between blood cholesterol and the occurence of CHD (Keys, 1970). In fact, no population has been reported to have a high rate of coronary heart disease and low blood cholesterol levels (NIH, 1985).

The Japanese population, for example, is characterized by a lower average cholesterol level than the United States and a lower frequency of coronary heart disease (NIH, 1985). At the other end of the continuum, the Finnish have a higher average cholesterol level and a higher rate of coronary heart disease than do U. S. citizens (NIH, 1985).

People who have migrated to another country with a higher average blood cholesterol level gradually acquire the blood cholesterol concentrations and coronary heart disease rates of their new country of residence (NIH, 1985). Japanese citizens, for example, who have migrated to Hawaii and to San Francisco have a higher cholesterol and higher risk of coronary heart disease than non-migrants (Robertson, 1977).

Genetic evidence suggests that severe coronary heart disease can result from high blood cholesterol levels in the absence of any other contributing risk factors (NIH, 1985). Research

has shown that children with inherited hypercholesterolemia, lacking the specific receptor that removes low-density lipoprotein-cholesterol (LDL-C) from the blood, suffer severe coronary heart disease and may die from a heart attack in childhood (Guyton, 1981). Further research on the disease arteries of these children has revealed large quantities of cholesterol in the plaque formations (Ross and Glomett, 1976).

Experimental animal studies conducted on rabbits, pigs, chickens and monkeys have found: (1) that many species develop atherosclerosis when fed diets that raise their blood cholesterol levels, (2) that hypercholesterolemic monkeys develop intimal lesions that progress from fatty streaks to raised plaques, paralleling similar atherosclerotic development in humans and (3) the atherosclerotic development can be reversed by lowering the dietary fat and cholesterol (Glueck and Connor, 1978; Guyton, 1981; NIH, 1985; Ross and Glomett, 1976).

Interestingly, Americans have begun changing their diets in recent years in response to advice from the medical, physical education and health related communitites. With this change in diet has come a drop in average blood cholesterol

levels and a drop in coronary heart disease incidence (NIH, 1985). It remains to be seen how much of this change is directly related to diet.

The Importance of High Density Lipoprotein-Cholesterol

In 1951, David Barr et al. at Cornell published a study showing that people with coronary heart disease had lower levels of high density lipoproteins than subjects without CHD (Barr et al., 1951). HDL's are subcomponents of total cholesterol; cholesterol is also made up of low-density lipoproteins (LDL's) and very low-density lipoproteins (VLDL's). HDL/s contain less cholesterol than LDL's and are thought to inhibit the action of LDL's in the development of atherosclerosis (Guyton, 1981).

The precise mechanism for HDL-C and its beneficial action is not known, but one leading theory suggests that HDL-C works against the atherosclerotic process by resisting the movement of LDL-C into the intima of a damaged arterial wall (Miller and Miller, 1975). A second theory suggests that HDL-C attaches to the forming lesion in the tissue and extracts the deposited cholesterol

promoting the regression of the fibrous plaque (Glomset, 1968).

The Cooperative Lipoprotein Phenotyping Study examined the relationship between coronary heart disease prevalence and fasting lipid levels (Castelli et al., 1977). Specifically, this case-controled study was an attempt to confirm Barr's (1951) early work and subsequent cross-sectional studies that showed an inverse relationship between HDL-C and coronary heart disease.

A total of 6,859 subjects, both male and female and of black, Japanese and white ancestry, were drawn from populations in Albany, Framingham, Evans County, Honolulu and San Francisco. All subjects were 40 years of age and older. In each major study group, mean levels of HDL-C were lower in persons with CHD than in those without the disease ($p. < .05$).

While LDL-C, total cholesterol and triglycerides were directly related to CHD prevalence, their relationship to CHD prevalence was less uniform in the various groups than the inverse HDL-C--CHD association (Castelli et al., 1977).

Based on the findings in the Framingham

Study, it appears that individuals can now be better diagnosed for CHD risk and treated earlier based on plasma HDL determinations, rather than using a simple total cholesterol rating (WDTB, 1979). The Framingham Study's results suggest that there is a direct linear inverse relationship between blood levels of HDL-C and the subsequent risk of CHD (Castelli et al., 1977).

Further research from the Framingham Study has demonstrated that the ratio of total cholesterol to HDL-C (TC/HDL-C) is a better predictor of CHD risk than any other combination and better than total cholesterol alone (WDTB, 1979).

Table 1 provides a composite summary of most of the important studies conducted since Barr's work in 1951 on the relationship between HDL-C and coronary heart disease (Heiss et al., 1980). There is a consistently strong inverse relationship between HDL-C and CHD prevalence across a wide variety of studies and methods. This relationship remains strong after controlling for total lipids, lipoprotein cholesterol fractions and nonlipid CHD risk factors (Heiss et al., 1980).

Table 1

Studies on High-Density Lipoprotein and Coronary Heart Disease

Author(s)	Date	Place	Findings
Barr et al.	1951	USA	HDL-C levels were lower in 22 patients with atherosclerosis than in matched controls
Nikkila	1953	Finland	HDL-C values were lower for 32 myocardial infarction patients than for matched controls
Jencks et al.	1956	USA	HDL-C levels were lower in 77 patients with atherosclerosis than in matched controls
Brunner and Lobl	1958	Israel	HDL-C levels were lower in 74 myocardial infarction patients than in matched controls
Gofman et al.	1966	USA	1961 men were followed for 10 years. HDL-C levels were lower in cases that developed CHD than in those who did not
Dodd and Mills	1969	England	HDL-C levels were lower in 19 myocaridal infarction survivors than in matched controls.
Wiklund et al.	1975	Sweden	Myocardial Infarction survivors (n=59) had lower HDL-C levels than matched controls
Berg and Borrensen	1976	Sweden	HDL-C levels were lower in 49 myocardial infarction patients than in 102 healthy men
Ononogbu	1976	England	HDL-C levels were lower in 50 ischemic heart disease patients than in 90 matched controls

Table 1 (continued)

Author(s)	Date	Place	Findings
Rhoad et al.	1976	Hawaii	HDL-C levels were higher in 1755 Japanese men without coronary heart disease than in 264 Japanese men with CHD manifestations
Castelli et al.	1977	USA and Hawaii	HDL-C values were lower for individuals with coronary heart disease than for in all study populations
Miller et al.	1977	Norway	21 CHD cases had lower HDL-C levels than 22 matched controls
Gordan et al.	1977	USA	A four-year follow-up of 2815 males and females yielded 142 incident cases of CHD. For each sex, HDL-C was independently and negatively associated with each major manifestation of CHD
Albers et al.	1978	USA	Myocardial infarction cases (n=90) had lower HDL-C levels than lipid-matched controls
Jenkins et al.	1978	Australia	The degree of angiographically determined coronary atherosclerosis was inversely associated with HDL-C in 41 males and females
Ishikawa et al.	1978	Norway	Twelve incident case of CHD had lower HDL-C levels than 16 matched controls
Noma et al.	1979	Japan	HDL-C levels were lower in 27 myocardial infarction patients than in controls

There is both strong biological and observational consistency in these studies (Table 1, pgs. 22-23). The association of HDL-C with the degree of CHD manifestation is consistently strong (Goldbourt and Medalie, 1979; Gordon et al., 1977; Jenkins et al., 1978).

In summary, the ratio of total cholesterol to HDL-C is now considered the strongest available predictor of CHD risk. The TC/HDL-C ratio conveniently summarizes an individual's CHD risk profile (Green et al., 1985).

Risk Factors Associated with Elevated Cholesterol and Coronary Heart Disease

A number of risk factors have been highly associated with the development of coronary heart disease, including, but not limited to, age, gender, hypertension, hyperlipidemia, diabetes mellitus and cigarette smoking (MRFIT, 1976). Of these factors, high blood pressure, smoking and lipid abnormalities are the three considered to be the most important (Castelli et al., 1977; Wilhelmsen, 1984). Most highly susceptible subjects have several risk factors (Castelli, 1984).

The Framingham Heart Study is an ongoing

study since 1949 involving 5,209 randomly selected men and women aged 30 to 62 years from the community of Framingham, Massachusets. It was designed with the intent of studying the influence of certain risk factors on the development of coronary heart disease (Castelli, 1984). Subjects are followed biennially with standardized examinations including a complete history, physical examination, chest radiography, electrocardiogram and various blood chemistry measures such as blood cholesterol, triglycerides, high density lipoprotein-cholesterol and blood glucose. Most (5,127) of the participants were asymptomatic at the time of entry.

Both age and gender emerged as clear risk factors. Every eighth man, aged 40 to 44 years of age at entry and free of coronary heart disease, developed some form of coronary heart disease within 14 years. Comparable rates for other age groups were every sixth man aged 45 to 49 years of age, every fifth man aged 50 to 54, and every fourth man 55 years of age or more (Castelli, 1984).

Women who had not yet experienced menopause had very low rates of CHD with only 11 subjects showing evidence of CHD while

still pre-menopausal from a possible 1600 candidates. Once past menopause, however, the rates for women increased dramatically. By the age of 60 years, in every fourth man and 17th women, some form of CHD had developed (Castelli, 1984).

Total cholesterol was found in the Framingham Study to be powerfully related to the subsequent development of CHD. Both LDL-C and VLDL-C were strongly related to increased incidence of CHD, while HDL-C was negatively associated with CHD. Individuals with TC/HDL-C ratios exceeding 4.5 were considered high risk for development of CHD. In the U. S., 50% of women and over 60% of men have ratios over 4.5 (Castelli, 1984).

Table 2 summarizes the Framingham data on the relationship between various TC/HDL-C ratios and subgroups tested in the Framingham Study.

A curvilinear relationship was found in the Framingham Study between blood pressure and subsequent development of coronary heart disease. This was true for both systolic and diastolic pressures. A systolic pressure of 160 mmHg (millimeters of Mecury) or greater

Table 2

Selected Groups According to Increasing Average Level of Total Cholesterol to HDL-Cholesterol

Group	TC/HDL-C
Vegetarians	2.9
Boston Marathon runners	3.5
Average among females without CHD	4.4
Average among males without CHD	5.1
Average among females with CHD	5.3
Average among males with CHD	5.8
Hypercholesterolemic females	6.0
Hypertriglyceridemic females	6.1
Hypertriglyceridemic males	6.9
Hypercholesterolemic males	7.3
Combined hyperlipidemic males	7.3
Combined hyperlipidemic females	8.4

Source: Castelli, W. "Epidemiology of Coronary Heart Disease: The Framingham Study." The American Journal of Medicine, February 27, 1984; 4-12.

or a diastolic pressure of 95 mmHg or greater increased the risk of CHD two-to-threefold, while arthothrombotic brain infarction was increased sevenfold at those levels (Castelli, 1984).

Other factors showing a strong relationship to the incidence of CHD were electrocardiographic abnormalities, blood glucose, weight, exercise, smoking, stress levels and a family history of CHD. Factors studied and found to be unrelated to CHD incidence were coffee, sleep, educational status, marital status, alcohol intake, percent greyness of hair, percent baldness and socioeconomic status (Castelli, 1984).

A number of studies have tested the theory that lowering blood cholesterol levels through dietary, secondary prevention or drug intervention subsequently lowers the incidence of coronary heart disease. Table 3 shows the drop in CHD incidence in several experimental groups versus controls associated with a drop in blood levels of cholesterol.

In every trial there was a decrease in subsequent rates of CHD proportional to the decrease in total cholesterol. With as low

Table 3

Percentage Fall in Coronary Heart Disease Rates in Various Cholesterol Lowering Trials

Study	Fall %
Diet	
Primary prevention:	
N. Y. Anticoronary Club	50
Helsinki Trial	50
Leren (Oslo)	44
Multiple Risk Factor Intervention Trial	7
Los Angeles Domicillary	25
Secondary prevention:	
Leren (Oslo)	35
Medical Research Council	17
Drugs	
Newcastle Trial	39
Scottish Society	19
Coronary Drug Trial	25
World Health Organization Trial	20
Krasnow (United Airlines)	65
Colestipol Trial	60
Gemifibrozil	73

Source: Castelli, W. "Epidemiology of Coronary Heart Disease: The Framingham Study." The American Journal of Medicine, February 27, 1984; 4-12.

as a one percent fall in cholesterol levels, there was a corresponding two to three percent drop in subsequent rates of CHD (Castelli, 1984).

Of all the factors indicated in CHD, the most important appears to be hyperlipidemia or, specifically, the levels of blood cholesterol and lipoproteins (Castelli, 1984). In Japan, for example, blood pressure levels and smoking are very high, but CHD has been rare because their blood levels of cholesterol have been low (Hagan et al., 1974).

Today, however, there has been a change in the average fat intake of the Japanese from 20 to 30 grams per day 20 years ago, to over 60 grams per day now (Robertson et al., 1977). With this increase in dietary fat has come an associated sharp rise in Japanese CHD rates (Robertson et al., 1977).

A vast body of literature supports the findings of the Framingham Study regarding the relationship between cholesterol and incidence of CHD. In reviews of epidemiological research concerning blood lipids and CHD, Fejfar and Masironi (1970) concluded that CHD is rare in individuals with blood cholesterol

below 200 mg/dl. If it does occur it is usually not related to atherosclerosis.

The relationship between diet and atherosclerosis has been known for some time. Almost 50 years ago, atherosclerosis was produced experimentally in rabbits by feeding them a diet rich in meat, milk and eggs (Anitschkow, 1933). Five years later, Anitschkow (1933) demonstrated that the atherosclerotic lesions produced in the animal were caused by the cholesterol and fat in the forced diet which produced hypercholesterolemia.

A number of reports have been published since 1953 showing significant associations between several dietary factors including cholesterol, saturated fat, percentage of fat in the diet, and total calories and coronary heart disease mortality rates (Brunner and Lobl, 1958; Christakis et al., 1966; Connor, 1961; Dayton et al., 1969; DeBakey, 1984; Ernst et al, 1980; Fejfar and Masironi, 1970; Glueck and Connor, 1978). In 1961, Connor reported a significant relationship between average daily cholesterol consumption and national coronary heart disease mortality rates for middle aged men.

These earlier findings and similar results from other published studies were confirmed in an extensive study published in 1977 by the Department of Community Health and Preventative Medicine from Northwestern Medical School in Chicago (Stamler, 1978). This study compared mortality data from 22 developed countries with data from the Food and Agriculture Organization on national food balances for 20 of those countries.

Significant associations between CHD mortality rates and several dietary factors including saturated fat and cholesterol were found in the Northwestern Study. Significant associations were also demonstrated between CHD mortality rates and cigarette smoking and automobiles per 1,000 population, the latter considered an indication of a sedentary lifestyle (Stamler, 1978).

A number of international studies have been conducted since 1956 comparing dietary levels of cholesterol and saturated fat to the degree of atherosclerotic development of the aorta and coronary arteries at autopsy (Kimura, 1956; McGill, 1968; Stamler, 1978). Kimura (1956) found a significant relationship

between the level of coronary and aortic involvement and the intake of saturated fat and cholesterol in the diet. This was also discovered in a follow-up study on dietary fat of 31,000 persons, aged 10 to 69, conducted by the International Atherosclerosis Project in 15 cities and countries (McGill, 1968).

The Seven Country Study compared dietary factors and atherosclerotic development in 1200 men, aged 40 to 59. Significant correlations were found between saturated fat in the diet, cholesterol in the diet and CHD rates, but not between total caloric intake nor sugar intake and CHD after controlling for saturated fat intake. (Keys, 1970). Both blood pressure and cigarette use were significantly related to CHD incidence.

Using coefficients for serum cholesterol, blood pressure and cigarette smoking obtained from the Framingham Study, Keys et al. found a significant high-order association between the five-year predicted and observed rates for the several populations (Stamler, 1978). In 1976, Keys et al. published a ten-year follow-up study showing that the essential interrelationships between dietary lipids,

serum cholesterol and CHD rates were the same at statistically significant levels (Stamler, 1978).

Other research has examined the relationship between emigrating to an area with a diet higher in in total fat, saturated fat and cholesterol and subsequent CHD rates (Hagan et al., 1974; Kato et al., 1973; Tillotson et al., 1973). Significant differences in diet composition, specifically in mean levels of total fat, saturated fat and cholesterol, were found between Japanese men who remained in Japan and those who had emigrated to the U. S. (Hagan et al., 1974; Kato et al., 1973; McGill, 1968; Tillotson et al., 1973).

Corresponding to these differences in nutritional patterns, mean serum cholesterol levels of Japanese men were 17% lower than for Hawaiian-Japanese men and 21% lower than for California-Japanese men (Hagan et al., 1974). Further research has demonstrated a significantly lower incidence of CHD mortality in the Japanese men when compared to their American counterparts. This was directly related to their lower mean serum cholesterol levels and independent of other risk factors

(Robertson et al., 1977).

Similar results have been demonstrated in domestic studies when the incidence of CHD has been examined in relationship to certain dietary factors, including dietary intake of cholesterol, saturated fats and total fat in the diet (PPRG, 1978).

Stamler (1978), in his review of national epidemiological studies, points out that U. S. epidemiologic studies yield only low-order or no correlations when the unit of measurement is the individual. He points out that most U. S. studies on dietary lipid intake and serum cholesterol are carried out on too few individuals. Also, too few measurements have been made on these individuals, leading to considerable intra-individual variation and, a lower correlation than would be expected on the basis of all other available evidence (Stamler, 1978).

Stamler suggests that at least nine days of food records are required to estimate the correlation between dietary lipid intake and serum cholesterol with reasonable precision and to avoid marked underestimation due to intra-individual variability (Stamler, 1978).

Overwhelming evidence, he points out, links dietary lipids to serum cholesterol levels and the association between serum cholesterol and CHD incidence is clear.

Research on other risk factors, including blood pressure and cigarette smoking, has been mentioned. Further research has indicated that cigarette smoking may significantly alter the TC/HDL-C ratio by lowering HDL-C levels (Criqui et al., 1980). Criqui et al. studied 2,663 men and 2,553 women, ages 20 to 69, in ten North American populations. Those men and women who were heavy smokers had significantly lower ($p. < .01$) HDL-C levels than lighter smokers. The association was dose-dependent, indicating a possible causal relationship between cigarette smoking and lower levels of HDL-C.

One other risk factor, alcohol consumption, has been negatively associated with incidence of CHD (Blackwolder et al., 1980; Camargo et al., 1985; Ernst et al., 1980; Gordon and Doyle, 1985; Hennekens et al., 1978; Kittner et al., 1983; Klatsky et al., 1974). However, available results are not all consistent and one recently published study (Gordon and Doyle

(Robertson et al., 1977).

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1985) has suggested caution in interpreting these results.

The Albany study, a large, case-controlled study on drinking and coronary heart disease, has been going on since 1953 (Gordon and Doyle, 1985). A total of 1910 men, aged 38 to 55 years, were examined once in 1953 and given follow-up exams in 1953-54 and 1971-72. In the initital period, there was no clear evidence of a relationship between the rate of alcohol consumption and CHD incidence. In the later period, men whose monthly consumption was 60 ounces or more had a lower than average CHD incidence rate. A negative relationship with drinking held for all manifestations of coronary heart disease.

Because of the absence of a relationship between drinking and CHD incidence in the initial follow-up of the Albany study, the researchers have suggested the need for caution on making any conclusions regarding the association between drinking and CHD risk. Also, because drinking is a matter of choice, confounding factors may influence the results of studies testing the relationship between drinking and CHD (Gordon and Doyle, 1985).

The Role of Exercise in Decreasing Coronary Heart Disease Risk

Exercise decreases coronary heart disease risk in a number of important ways. Both systolic blood pressure and circulating levels of catecholamines are reduced at rest and during exercise as a result of endurance training (Hartley et al., 1972).

High levels of catecholamines and high blood pressure are both associated with increased CHD risk (Castelli, 1985; Hartley, 1985). Also important, exercise encourages collateral formation in coronary and peripheral arterial beds and has been demonstrated to increase the cross-sectional area of the coronary arterial network (DeBakey, 1984; Guyton, 1981).

In the Framingham Study, the association between exercise and coronary heart disease was examined by administering questionnaires at the time of entry (Kannel et al., 1971). Based upon responses, individuals were categorized for activity habits into one of three groups: least, intermediate and most active. After ten years, follow-up results revealed significant differences between the incidence

of coronary heart disease and the level of physical activity at entry.

The moderate exercise group had a significantly lower incidence of CHD than the least active group, while no difference was observed between the moderate and heavy exercise groups. This suggests that there is no additional protection afforded an individual who exercises above moderate levels.

A threshold value for exercise has been estimated by the Framingham researchers based on the results of their study. This level is the equivalent of eight MET-hours per week, or the equivalent of eight hours of brisk walking a week (Hartley et al., 1972).

This suggests that an individual could achieve maximum protection against heart disease by walking eight hours a week or by substituting more strenous activities (running, swimming, tennis) in less time. This is unlike the direct linear, dose-response correlation seen between dietary factors, serum cholesterol levels and increased risk of coronary heart disease (Castelli, 1984).

Paffenberger et al. (1978) studied 16,936 male college alumni to test the relationship

between energy expenditure and the risk of a first heart attack. Individuals who reported climbing fewer than five flights of stairs per day were at 25% increased risk of heart attack compared with those who climbed more than five. Individuals who walked fewer than five city blocks had 23% more heart attacks than those who walked more. Men who reported not playing any sports or only light sports had a 38% greater risk of heart attack than those who engaged in vigorous activities.

Based on the results of the Paffenberger study, individuals exercising less than three hours a week or participating in exercise that consumed less than 2,000 kcal/week, had a 64% greater risk of having a heart attack than those who were more active.

Other studies conducted on postal workers (Kahn, 1963; Morris et al., 1966), railroad workers (Taylor et al., 1962, 1970), policemen and firefighters (Peters et al., 1983) and civil servants (Morris et al., 1980) have reported significantly lower rates of CHD in the more physically active men.

In light of the available research, there is little doubt that exercise can help lower

the risk of coronary heart disease. The relative amount or threshold value needed to obtained the lower risk benefits has been estimated at about 2,000 kcal/week (Castelli, 1984; Paffenberger et al., 1978). Exercise apparently helps elevate blood HDL-C levels, while helping to lower the cholesterol-rich LDL-C levels, thereby favorably altering the TC/HDL-C ratio and reducing the risk of CHD (Castelli, 1984).

In a recent study by Tran and Weltman (1985), ninety-five studies conducted between September of 1955 and October of 1983 measuring changes in human lipid and lipoprotein levels, were analyzed using meta-analysis. This study examined the effects of weight loss associated with the effects of exercise on serum lipid and lipoprotein levels.

In all cases, there was a significant drop in cholesterol levels, expecially LDL-C levels, in response to exercise. The reductions in LDL-C and total choelsterol were greatest when exercise was combined with weight loss. Favorable decreases in both total cholesterol and in LDL-C levels have also been reported in other studies (Altekruse and Wilmore, 1973; Cowan, 1983; Haskell, 1984; Lopez et al.,

1974; Peltonen et al., 1981; Rotkis et al., 1982).

Exercise has consistently been associated with higher HDL-C concentrations (Haskell, 1984). Cook et al. (1984) found that families that participated regularly in exercise also had higher levels of HDL-C. Adner and Castelli (1980) demonstrated that distance runners had significantly higher HDL-C levels than non-running controls, even after controlling for relative weight and triglyceride levels. Haskell (1984) found the relationship between exercise and increased HDL-C levels was consistent across a wide variety of studies.

Cross-sectional studies have also shown significantly higher HDL-C values for endurance trained athletes versus sedentary controls (Hagan and Gettman, 1983; Rotkis et al., 1980; Wood and Haskell, 1979).

Garay and Butterfield (1984) examined the effects of exercise on HDL-C levels in competitive and recreational swimmers versus non-swimming controls. After controlling for diet and alcohol consumption, HDL-C values were found to be significantly higher in both the recreational and competitive swimmers.

versus the controls.

Hartung and Squires (1980) obtained similar results in comparing 147 male runners and 78 sedentary controls. HDL-C values were significantly higher for both high and low-milage runners when compared to non-running controls. Rotkis (1980) obtained similar results in a runners versus controls study of 109 subjects after controlling for body composition, diet, age and family history of CHD.

Farrell and Barboriak (1980) found significant increases in HDL-C levels associated with endurance training when examining the effects of exercise on 16 previously sedentary subjects. These changes were independently associated with exercise after controlling for diet, smoking, alcohol consumption and body weight.

A dose-response relationship between amount of training and plasma HDL-C levels exists, but this relationship tails off near higher levels of activity (Farrell and Barboriak, 1980; Hartung and Squires, 1980; Hartung et al., 1980; Lehtonen and Viikari, 1978; Rotkis et al., 1982; Wood et al, 1976; Wood et al.,

exercise needed to obtain the benefit of decreased risk of CHD appears to have a threshold value of about 2,000 kcal per week, above which there is no added benefit.

Tracking Studies: The Importance of Identifying Risk Patterns at an Early Age

Many researchers have questioned the need for developing programs for CHD prevention at early ages and in populations below the 95th percentile in blood cholesterol levels (Kronmal, 1985; NIH, 1985; Rahimtoola, 1985). They have asked for more evidence linking cholesterol to CHD in younger populations. They contend that unless a direct link can be drawn between cholesterol levels and CHD development in these younger populations, it is irresponsible for the medical community to suggest these individuals lower their dietary cholesterol and adjust other risk factors.

Recently, a number of studies have addressed these arguments and the results have been overwhelmingly in favor of intervention measures and prevention of CHD in early life (Beaglehole et al., 1980; Freedman et al.,

1985; Mellies et al., 1985; Newman et al., 1986; Orchard et al., 1983; Radhakrishnamurthy et al., 1985; Thorland and Thomas, 1981).

Others have also suggested the need for further examination of risk factor patterns in younger populations and the need to develop CHD prevention programs, even as early as the elementary school level (Berg et al., 1983; Harlan and Stross, 1985; Masironi, 1978).

Newman et al., (1986) assessed the relationship of risk factors for cardiovascular disease to early atherosclerotic lesions in the aorta and coronary arteries in 35 individuals (mean age at death = 18 years). Aortic fatty streaks were stongly related to antemortem levels of both total and low-density lipoprotein-cholesterol ($r=0.67$, $p. < 0.0001$ for each association), independent of race, sex and age, and were inversely correlated with the ratio of HDL-C, LDL-C and VLDL-C ($r=-0.35$, $p. < 0.06$).

The authors of the Newman study have suggested that these results demonstrate the importance of risk factor levels to early anatomical changes in the aorta and coronary arteries.

Mellies and others (1985) followed HDL-C and LDL-C blood levels of 77 children and 53 adults from a single large family line with a family history of hypercholesterolemia from 1973 to 1984. The rank correlations between the 1973 and 1984 measurements for LDL-C were 0.73, 0.74 and 0.87, and for HDL-C were 0.55, 0.73 and 0.65 ($p. < 0.0001$ for all correlations), respectively for individuals who were less than 12, 13 to 19 and less than 20 years old in 1973.

LDL-C and HDL-C levels in childhood were highly predictive of adult values in the Mellies study, emphasizing the importance of evaluation of risk factors for CHD at an early age.

Since it is now recognized that atherosclerosis begins in early childhood (Guyton, 1981; Newman et al., 1986; NIH, 1985; WDTB, 1979), and that this early atherosclerotic development is highly correlated with blood lipid and lipoprotein levels (Newman et al., 1986), it seems reasonable to conclude that identification of early factors leading to elevated blood cholesterol levels and modification of those factors may prevent subsequent

atherosclerotic development. Studies designed to test this hypothesis have not yet been published.

Summary

A body of evidence linking elevated blood cholesterol levels and various associated risk factors to coronary heart disease has been reviewed. Tremendous support for a causal role of cholesterol in atherogenesis has been presented, including clinical intervention studies, epidemiological studies, genetic studies and experimental research. These data establish beyond any reasonable doubt the close relationship between total blood cholesterol and coronary heart disease.

Clinical trials, such as the LRC-CPPT (1984), have successfully demonstrated that a reduction in blood choelsterol levels will significantly reduce the risk of developing coronary heart disease. Overall, these clinical trials have demonstrated that each 1% drop in blood cholesterol levels yields a 2% drop in CHD rates (NIH, 1985).

The role of HDL-C as a beneficial lipoprotein in the prevention of coronary heart

disease has been extensively discussed. It has been shown, base on evidence from the Framingham Study (1977) and other studies reviewed, that using the ratio of TC/HDL-C is a better predictor of CHD risk than using cholesterol alone.

Consistently, HDL-C has demonstrated a strong inverse association to CHD risk. Possible reasons for this beneficial effect may include the efflux of cholesterol away from the affected sites (Glomset, 1968) or the inhibition of LDL-C attachment to receptor sites (Miller and Miller, 1975).

Various risk factors associated with CHD include age, gender, hypertension, smoking and hyperlipidemia. Based on the results of several international studies, it has been determined that hyperlipidemia and causative dietary factors are the leading promoters of CHD (Castelli, 1984).

The development of CHD was shown to be even more highly correlated with individuals who had two or more risk factors, illustrating the multifactorial relationship of CHD to risk factors. Cross-cultural studies, however, have illustrated that dietary factors, including

intake of cholesterol, saturated fat and total dietary fat were more important than other risk factors, including smoking and hypertension.

Alcohol consumption was shown to be negatively correlated with CHD in some studies and not associated in others. Conclusions about the possible HDL-C raising effect of alcohol have been guarded.

Exercise is consistently associated with increased HDL-C levels and lowered incidence of coronary heart disease. There appears to be a threshold value for exercise of about 2,000 kilocalories per week, beyond which greater protection against CHD is not afforded.

Endurance trained individuals consistently demonstrated lower cholesterol and higher HDL-C levels than their non-exercising counterparts. At least one study (Herbert et al., 1984) has shown that the elevated HDL-C levels may be due to prolonged HDL-C survival time, rather than increased production of HDL lipoproteins.

Recently published tracking studies, which are studies that follow populations over a period of several years or more to test the level

of association between blood choelsterol levels and CHD incidence, demonstrated a strong relationship between blood characteristics at an early age and the accelerated development of CHD. These findings have been confirmed by the demonstration of very strong relationships between early cholesterol/HDL-C ratios and the degree of CHD progression at death as determined by autopsy (Newman et al., 1986).

In summary, the review of the literature yielded the following generalizations:

1. A causal role for cholesterol in the development of atherosclerosis has been established and this role is independent of other risk factors.

2. HDL-C has been shown to have a favorable effect on lowering CHD risk, making the ratio of TC/HDL-C the most effective predictor of CHD risk.

3. A number of important risk factors are associated with CHD development, the most important of which are dietary factors.

4. Exercise has been shown effective in favorably decreasing the TC/HDL-C ratio by increasing HDL-C levels.

5. Tracking studies demonstrating the role of blood cholesterol levels in early atherosclerotic development illustrate the need to examine factors associated with poor TC/HDL-C ratios and the development of preventative programs even at early ages.

CHAPTER III

Research Methodology

The purpose of this study was to determine the relationship of exercise and diet in predicting the total cholesterol/high density lipoprotein-cholesterol ratio in college age males and females.

The Sources of Data

The subjects in this study were 22 female and 12 male students, aged 18 to 23 years, taken from the University of the Pacific's Heart, Exercise and Nutrition class of the Spring, 1986 semester. The 34 subjects were the remaining valid cases from 55 possible students, after controlling for age, medication use, contraceptive use, hormone use, smoking and intense physical activity prior to testing.

Data Collecting Instrument

Individual dietary consumption was measured by three-day dietary records (Appendix A) designed by Nutri-Calc systems. Validity and reliability of the instrument were determined

in an analysis and comparison of both dietary collection methods and the actual assesment tool used to collect the data (Morgan et al., 1978; Burger and Suitor, 1984).

The use of a three-day dietary analysis was found to be more representative of an actual dietary pattern than a one day record. The three-day record showed high reliability (r=.83) with actual dietary history (Burger and Suitor, 1984).

Validity of the dietary recall program was assesed in a comparison of available programs against the recommended program of the United States Department of Agriculture (Handbook No. 456). Nutri-Calc coefficients for validity were high (r=.90) on nearly all items (Burger and Suitor, 1984).

Intensity, duration and frequency of physical activity was modeled on the Minnesota Leisure Time Physical Activity Questionnaire, as established by Taylor et al. (1978). The form used is presented in Appendix B.

Taylor et al. (1978) assessed the validity and reliability of the instrument by comparing the calculated work in kcal/min/kg to actual performance on a graded exercise test. A

clear and statistically significant (r=.52) relationship between reported activity levels and exercise performance was established for each exercise endpoint examined.

Blood samples were measured at Lodi Memorial Hospital by a qualified lab technician using standard enzymatic techniques as outlined in the Worthington Diagnostics Technical Bulletin (1978). The enzymatic method of analyzing plasma samples in determining lipid and HDL-C concentrations is an accurate, commonly used technique (Haskell, 1984).

Procedures for Data Collection

Three-day dietary record sheets were distributed to the subjects in class five days prior to testing and a professional dietary expert gave instructions on how to properly complete a dietary record. Each student also received a 30-page food coding sheet with proper recording instructions (Appendix A).

Each student was also given written instructions on how to complete all forms with an attached questionnaire (Appendix C) on control items for the study.

At the time all forms were distributed (Thursday, January 16, 1986), the students also signed up for blood testing and dietary recall interviews, which were conducted on Tuesday, January 21, 1986. Students were instructed to record food and activity for Thursday, Friday and Saturday, January 16-18. Each student was also instructed to fast at least 12 hours prior to testing and to refrain from intense physical activity for at least three days prior to testing.

Students began testing at 9:00 a.m. on Tuesday, January 21, 1986 at the Cowell Student Health Center at University of the Pacific. Five-milliliter blood samples were drawn by nurses at the facility into vacutainer tubes containing heparin to prevent clotting of the blood. These samples were refrigerated for analysis later in the week.

As each sample was drawn, the sample was recorded with the subject's name and the student was sent to the Cowell conference room for his/her interview. Both the dietary expert and the researcher conducted the interviews. Both were previously trained in conducting dietary interviews.

All females were interviewed by the registered dietician, while the males were interviewed by the researcher. Dietary and physical activity records were reviewed for accuracy and clarity and control questions were also double checked. Follow-up interviews were given on Wednesday, January 22, 1986 and Thursday, January 23, 1986 for individuals who had missed their appointment (n=4) or incorrectly filled out the forms (n=6).

Blood samples were taken to Lodi Memorial Hospital for analysis. Total cholesterol and HDL-cholesterol levels were determined as described and the TC/HDL-C ratios were calculated from this data.

Dietary records were taken to St. Joseph's Hospital in Stockton to be calculated on the Nutri-Calc program as described. Physical activity information was computed by hand and subjects were classified as either (1) no or light activity of up to 200 kilocalories per day, (2) moderate activity of up to 1,000 kilocalories per day or (3) high activity of above 1,001 kilocalories per day (Paffenberger et al., 1982).

Analysis of the Data

Data obtained from the analysis of the blood samples, dietary values, physical activity values and control values were considered for entry into data files on the Vax computer system at the University of the Pacific.

All subjects with positive answers to control questions (Appendix C) were excluded from the study at this point (n=21).

Data files were than set up for total subjects, males and females. A forward stepwise multiple regression analysis was then set up using the Statistical Package for the Social Sciences analysis program. Correlations, partial correlations, coefficients of determination (r^2) and r^2 change were then examined for linearity and association on each of the variables as specified in the hypotheses.

A level of $p. < .05$ was selected as the level to demonstrate a significant linear relationship.

CHAPTER IV

Results, Summary, Conclusions

Results

The main problem of this study was to determine the relationship of exercise and diet in predicting the total cholesterol/high density lipoprotein-cholesterol ratio in college age males and females. The hypothesis for this problem was that there would not be a linear relationship between exercise and diet and the TC/HDL-C ratio.

Results of the analysis, including the descriptive data for the population and the relationship of the dietary variables cholesterol intake (CHOL), unsaturated/saturated fat ratio (SAT), percentage of dietary fat (FAT) and physical activity (PA) to the TC/HDL-C ratio (RATIO) are presented in tables 4 and 5 respectively. The raw data can be found in Appendix D.

The coefficient for determination (r^2) showed only a small (.1453) and non-significant relationship between the variables tested and the TC/HDL-C ratio. This low relationship was probably due to the tremendous variability

Table 4

Descriptive Data for the Population

Variable	Mean	1 SD	Label
RATIO	3.014	± .576	TC/HDL-C ratio
CHOL	363.000	± 474.342	Dietary cholesterol
SAT	.400	± .203	Polyunsaturated/saturated fat ratio
FAT	36.353	± 6.559	Percentage of dietary fat
PA	2.147	± .892	Physical Activity

Table 5

Relationship of the Dietary Variables Cholesterol Intake (CHOL), Unsaturated/saturated Fat Ratio (SAT), Percentage of Dietary Fat (FAT) and Physical Activity (PA) to the Ratio TC/HDL-C (RATIO)

Variable	Mult r	r^2	$r^2\Delta$	Beta in	Correl.
CHOL	.3737	.1397	.1397	.3737	.3737
SAT	-	-	-	.0575	.0334
FAT	-	-	-	.0004	.0766
PA	.3812	.1453	.0057	-.0517	.0690

Dependent variable = RATIO n=34

in the variable cholesterol intake in which the standard deviation (474.342) was larger than the mean (363.000), creating a skewed distribution.

Thus, the first hypothesis of this study, that there would not be a linear relationship between exercise and diet and the TC/HDL-C ratio was accepted.

The first subproblem of this study was to determine the relative importance of frequency, duration and intensity of exercise, after controlling for all other factors, in predicting the TC/HDL-C ratio in college age males and females. The hypothesis for this problem was that there would not be a linear relationship between frequency, duration and intensity of exercise and the TC/HDL-C ratio after controlling for all other factors.

Table 5 (p. 61) contains the data that addresses this hypothesis. The ability of physical activity to predict a change in the TC/HDL-C ratio was so low that it did not meet the entering requirements (probability of F ratio = .500 or greater to enter) as established in the problem criteria. Therefore, physical activity was not even considered

in the equation.

Thus, the second hypothesis of this study, that there would not be a linear relationship between exercise and the TC/HDL-C ratio, was also accepted.

The second subproblem of this study was to determine the relative importance of the percentage of total fat in the diet (after controlling for all other variables) in predicting the TC/HDL-C ratio in college age males and females. The hypothesis for this problem was that there would not be a linear relationship between the percentage of total fat in the diet and the TC/HDL-C ratio.

Table 5 (p.61) contains the data that addresses this hypothesis. The ability of the variable percentage of fat in the diet to predict a change in the TC/HDL-C ratio was again too low to meet the requirements set up in the equation.

Thus, the third hypothesis of the study, that there would not be a linear relationship between the percentage of fat in the diet and the TC/HDL-C ratio was accepted.

The third subproblem of this study was to determine the relative importance of the

ratio of polyunsaturated to saturated fats in the diet (after controlling for all other variables) in predicting the TC/HDL-C ratio in college age males and females. The third hypothesis tested for this subproblem was that there would not be a linear relationship between the ratio of polyunsaturated to saturated fats in the diet (after controlling for all other variables) and the TC/HDL-C ratio.

Table 5 (p. 61) contains the data that addresses this hypothesis. Again, the ability of the variable polyunsaturated/saturated fat in the diet to predict a change in the TC/HDL-C ratio was so low that it could not meet the requirements of the equation.

Thus, the third hypothesis of this study, that there would not be a relationship between the polyunsaturated/saturated fat ratio in the diet and the TC/HDL-C raio was accepted.

The fourth subproblem of this study was to determine the relative importance of the amount of cholesterol in the diet (after controlling for all other variables) in predicting the TC/HDL-C ratio in college age males and females. The hypothesis tested for this

problem was that there would not be a linear relationship between the amount of cholesterol in the diet and the TC/HDL-C ratio.

Tables 5 (p. 61) and 6 contain the data that addresses this hypothesis. The variable cholesterol showed a much higher correlation ($r = .37372$) than any other variable with the TC/HDL-C ratio. When squared, cholesterol in the diet yielded a coefficient of determination (r^2) of .13967, meaning that after controlling the other variables in the equation, the variable CHOL predicted 14% of the change in the TC/HDL-C ratio. This correlation was significant at the .05 level (.0295).

Thus, the fourth hypothesis of this study, that there would not be a linear relationship between dietary cholesterol and the TC/HDL-C ratio, was rejected.

The fifth and final subproblem of this study was to determine if any of the above interactions between the exercise and dietary variables and the TC/HDL-C ratio were different for males than for females in this college age population. The hypothesis tested for this problem was that there would not be any significant differences between the ability

Table 6

Multiple Stepwise Regression Equation for Dietary Cholesterol

Dependent variable: RATIO

Control Variables:

Saturated Fat in Diet
Percentage of Fat in Diet
Physical Activity

Multiple regression: stepwise

Multiple R:	.37372
R^2:	.13967
R^2 change:	.13967
Beta:	.373719

Significant at .0295

of any variable or combination of variables to predict the TC/HDL-C ratio in males versus females.

Tables 7 and 8 contain the data that describes the male and female populations and tables 9 and 10 contain the data that addresses this hypothesis.

As shown in table 9, cholesterol had a very high linear relationship with the TC/HDL-C ratio (r^2 = .3337). However, due to the few subjects available in this population (n=12), this relationship was not significant at the .05 level. The total r^2 change for the remaining three variables, SAT, FAT and PA was less than .04 and did not yield any significant additional information to the ability of these factors to predict the TC/HDL-C ratio.

As seen in table 10, the variables involved in the equation for the female population all showed at least a minimum association with the dependent variable TC/HDL-C ratio. Only dietary cholesterol, however, showed a significant relationship ($p. < .05$) with the dependent variable.

The variable polyunsaturated/saturated fat yielded additional information to the

Table 7

Descriptive Data for Male Population

Variable	Mean	1 SD	Label
RATIO	3.118	±.508	TC/HDL-C ratio
CHOL	603.917	±741.287	Dietary cholesterol
SAT	.333	±.187	Polyunsaturated/ saturated fat ratio
FAT	36.250	±5.594	Percentage of fat in the diet
PA	2.5000	±.798	Physical Activity
			(n=12)

Table 8

Descriptive Data for Female Population

Variable	Mean	1 SD	Label
RATIO	2.958	±.614	TC/HDL-C ratio
CHOL	231.591	±120.326	Dietary cholesterol
SAT	.436	±.206	Polyunsat/ sat. fat ratio
FAT	36.409	±7.156	Percentage of fat in diet
PA	1.955	±.899	Physical Activity
(n=22)			

Table 9

Summary of Multiple Regression Analysis
for all Variables: Males

Variable	MultR	R^2	$R^2\Delta$	BetaIn	Correl.
CHOL	.5777	.3337	.3337	.5777	.5777
SAT	-	-	-	-.1798	-.0697
PA	-	-	-	.0449	.2256
FAT	.6068	.3682	.0345	-.0443	.2486

Dependent variable = RATIO (n=12)

Table 10

Summary of Multiple Regression Analysis
for all Variables: Females

Variable	MultR	R^2	$R^2\Delta$	BetaIn	Correl.
CHOL	.3332	.1110	.1110	.3332	.3332
SAT	.4201	.1765	.0654	.2728	.1241
FAT	.4604	.2120	.0356	-.2124	.0112
PA	.4755	.2261	.0141	.1285	.0330

Dependent variable = RATIO (n=22)

equation ($r^2 \Delta = .0654$), as did the variables percentage of fat in the diet ($r^2 \Delta = .0356$) and physical activity ($r^2 \Delta = .0140$). The total predictability of all variables ($r^2 = .2261$) was significant at the .05 level.

In comparing the results of the analysis between males and females, a significant difference was not found. Thus, the fifth hypothesis, that there would not be a significant difference between the ability of any variable or combination of variables to predict the TC/HDL-C ratios differently in the male population when compared to the female population, was accepted.

Summary

The purpose of this study was to determine the relationship of exercise and diet in predicting the total cholesterol/high density lipoprotein-cholesterol (TC/HDL-C) ratio in college age males and females.

The hypotheses were state in null form as follows:

1. There would not be a linear relationship between exercise and diet and the TC/HDL-C

ratio.

2. There would not be a linear relationship between frequency, duration and intensity of exercise (after controlling for all other factors) and the TC/HDL-C ratio.

3. There would not be a linear relationship between the percentage of total fat in the diet (after controlling all other variables) and the TC/HDL-C ratio.

4. There would not be a linear relationship between the ratio of polyunsaturated to saturated fats in the diet (after controlling for all other variables) and the TC/HDL-C ratio.

5. There would not be a linear relationship between the amount of cholesterol in the diet (after controlling for all other variables) and the TC/HDL-C ratio.

6. There would not be a significant difference between the ability of any variable or combination of variables to predict the TC/HDL-C ratio in college age males versus females.

The subjects in this study were 22 females and 12 males, ages 18 to 23, taken from the University of the Pacific's Heart, Exercise and Nutrition class of the Spring, 1986 semester.

The 34 subjects were the remaining valid cases from 55 possible subjects, after controlling for age, medication use, contraceptive use, hormone use, smoking and intense physical activity prior to testing.

Individual dietary consumption was measured by three-day dietary records designed by Nutri-Calc systems and checked through dietary record interviews. Physical Activity was measured using an adaptation of the Minnesota Leisure Time Physical Activity Questionnaire and checked by follow-up interview. Blood samples were analyzed by a qualified lab technician at Lodi Memorial Hospital using standardized enzymatic techniques.

Data was analyzed in a forward stepwise multiple regression equation based on procedures described in the Statistical Package for the Social Sciences (SPSS-X) manual (1985) for data analysis on the Vax computer system at University of the Pacific's computer laboratory.

Results of the data analysis indicate that there was not a significant linear relationship between exercise and diet and the TC/HDL-C ratio.

Cholesterol was, however, associated with

the TC/HDL-C ratio and this proved to be a significant relationship ($p. < .05$) after controlling for all other variables in the equation.

No significant difference was found for the ability of any variable or combination of variables to predict the TC/HDL-C ratio in males versus females.

Conclusions

Based on the results of this study, the following conclusions were formed:

1. Exercise and dietary variables have only a limited ability to predict TC/HDL-C ratios in this college age population of males and females.

2. Frequency, duration and intensity of exercise, as reported through questionnaire interview, has no predictive power towards the TC/HDL-C ratios in this college age population of males and females.

3. The percentage of total fat in the diet cannot be used as a reliable predictor of TC/HDL-C ratios in this college age population.

4. The ratio of polyunsaturated to saturated fats in the diet cannot be used as a reliable

predictor of the TC/HDL-C ratio in this college age population.

5. The amount of cholesterol in the diet does have a significant linear relationship to TC/HDL-C ratios in this college age population and may demonstrate some predictive power towards those ratios.

6. Although some differences in the relative importance of dietary cholesterol and the other variables to the TC/HDL-C ratio showed up in the male versus female analysis, these differences were not significant and may be more a reflection of the different sample sizes than any difference between the two populations.

Discussion

A substantial body of literature supports the importance of studying younger populations to determine the factors involved in the begining of coronary heart disease development. Among the leading factors implicated in the development of atherosclerosis are inactivity, high blood levels of cholesterol, high dietary intake of cholesterol, dietary saturated fat, total dietary fat and gender-related differences.

This study examined the association between these dietary and exercise factors and the TC/HDL-C ratios in a college age population. The TC/HDL-C ratio has been established as the best indicator of future coronary heart disease risk (Castelli et al., 1977).

The results of this study showed non-significant correlations between exercise, saturated fat/unsaturated fat ratio, total dietary fat and gender differences and the TC/HDL-C ratio in this population. The findings of this study are not consistent with the findings reviewed in the survey of the literature.

Foremost among the possible reasons for this inconsistency are the following: (1) there were too few subjects in the population (n=34) to dilute the skewed distribution caused by individual variation, (2) this population was atypical of former populations studied and little extrapolation can be made from one population to the other or (3) the variables considered in this study, although significant in older populations with higher blood levels of cholesterol, are not significant in predicting TC/HDL-C ratios in younger populations.

If the first suggestion is correct, that there were simply too few subjects in this study to account for individual variation, then further research on a similar, but larger population might lead to significant relationships between the considered variables and the TC/HDL-C ratio. However, the correlations between the individual variables and the TC/HDL-C ratio were so low (Table 5, p. 61), that even that projection seems doubtful.

Dilution appears to have an effect on the significant relationship established between dietary intake of cholesterol and the TC/HDL-C ratio. When one individual, whose dietary intake of cholesterol was more than seven standard deviations away from the mean, was removed from the study, the ability of dietary cholesterol to predict the TC/HDL-C ratio dropped from .1397 (Table 5, p. 61) to .0152. The tremendous difference in the two results caused by the dropping of a single individual illustrates the need for larger numbers in a study of this kind.

The second suggestion, that this population is atypical of other populations reviewed in the survey of the literature, appears to be correct. Most of the studies cited in the review of the literature used subjects who were

between the ages of 40 and 60 and had TC/HDL-C ratios above 4.5 (Castelli et al., 1977; LRC-CPPT, 1985).

The average TC/HDL-C ratio of this population was 3.014 (Table 4, p. 60). This is far below the average ratio of those tested in other studies and is only one-half the risk of the normal risk for CHD (WDTB, 1979). Not one individual in the final accepted population for study had a ratio as high as 4.5 (Appendix D).

It may also be that this population is unlike other similarly aged college populations. Thus, as suggested by Kolata (1985) and Kronmal (1985), it is probably incorrect and irresponsible to extrapolate from one select population to the general or other select populations.

As little research has been done on college-aged populations, it is difficult to compare the results of this study with any other findings. Thus, caution must be taken in extrapolating these results to individuals outside this special study group.

Because this population is probably atypical of other populations, the results of this study only suggest where further lines of research may be directed.

The third suggestion to explain the inconsistent findings is that the variables considered in this study may have a different effect on the TC/HDL-C ratio in a younger population than they do in an older population. It may be that these variables interact differently in younger individuals than they do in older individuals.

It is known that changes take place in individuals as they get older, leading to higher TC/HDL-C ratios in later life (Castelli et al., 1977). To what extent do the variables considered in this study influence those changes? This question deserves further research.

The basic purpose of this study was to determine to what extent each of the variables considered, including physical activity, dietary cholesterol, dietary fat, dietary saturated fat and gender, influenced the TC/HDL-C ratios in this select population. It was discovered that diet and exercise accounted for very little of the TC/HDL-C ratio in this population. Whether this was caused by the small sample size, the atypical population,

different variable effect or some unknown factor is undiscernable.

Of all the variables considered in this study, dietary cholesterol seemed to have the highest association with the TC/HDL-C ratio. This finding, however, must be tempered with the knowledge that cholesterol loses much of its predictive ability when a single case with an abnormal intake of cholesterol is taken out of the equation.

Possibly in response to a public health campaign to reduce dietary fat and cholesterol, serum cholesterol values of the general population in the U. S. have declined about 15 mg/dl for each age group despite an increase in body weight over the past 25 years (Harlan and Stross, 1985). Per capita consumption of eggs has dropped 26% since since 1950, while consumption of milk fat solids and lard has declined 39% and 79% respectively since 1940 (Stamler, 1978).

The per capita consumption of cigarettes has declined 25% since 1964 and the detection and treatment of hypertensives has increased 300% since 1969 (Stamler, 1978). It seems reasonable to conclude, therefore, that the American public is responding to advice about

preventative measures towards coronary heart disease.

The important question, is has there been a drop in CHD mortality rates in this country. The answer is a convincing yes. Since 1968, coronary heart disease has declined 19% for white males, 24% for white females, 29% for non-white males and 35% for non-white females (Stamler, 1978).

Despite these recent trends, CHD in the U. S. is still the leading cause of death and the rate of CHD incidence in the U. S. is still among the highest in the world. If the recent changes in diet, exercise and preventative health measures are responsible for the decline in CHD rates, then further research along these lines may help in promoting greater reductions in CHD in the future.

Several studies (Table 3, p. 29) have demonstrated significant reductions in CHD incidence through dietary modification and drug intervention. Based on the results of this study, this researcher must concur with the argument offered by Kolata (1985) and Kronmal (1985) that is may be wrong to extrapolate the findings of these older

hypercholesterolemic male studies to the general population.

However, it is difficult to argue against advice on dietary modification and risk factor reduction. Thus, this researcher sees a need to continue research efforts on a wider variety of subject populations, while continuing efforts towards providing sound preventative advice.

Recommendations

Based on the results of this study, the following recommendations are made:

1. A similar study should be done on a larger sample size, or specifically on males or females, to determine if these results differ significantly from the results of a larger study.

2. Further research should be conducted to delineate variables not considered in this study, including, but not limited to, smoking, hypertension, contraceptive use, medication use and alcohol consumption and their relative influence on the TC/HDL-C ratios.

Further research should be conducted on a wider variety of subject populations to form

a wider data base from which to draw inferences towards the general population.

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APPENDIX A

Personalized Nutrient Analysis Food Record

Instructions for completing a three-day food record:

1. Record all food eaten for three days that represents a typical diet pattern.

2. Be as accurate as possible in the recording of the type and amount of each food eaten. Weigh food, if possible. List amount in cups, tablespoons, ounces, etc.... Raw fruit may be indicated by size and/or piece (i. e., small apple).

3. It is recommended to write down the food and/or beverages as it is consumed. It is not necessary to list diet sodas, tea or coffee (unless milk or sugar is added).

4. The accuracy of your results depends upon how precise and inclusive you can be. For example:

1-16/10 am/Lunch:

Tuna sandwich:

wheat bread	2 slices
tuna (waterpack)	2 ounces
mayonnaise	1 tbl.

Salad:

lettuce	1 cup
dressing, Italian	1 tbl.

APPENDIX A (continued)

5. Find the food on the provided list and record servings and code number.

6. Next, use the form on this page to total your food record.

7. For homemade mixed dishes, break down the dish into food components as shown below:

Example:

Chicken casserole:

½ cup noodles
¼ cup cheese
2 ounces chicken

Date/Time/Meal	Description of Food	Servings

APPENDIX B

Physical Activity Record

Date/Time	Description of Activity	Duration

APPENDIX C

Control Questionnaire

For the purposes of this study, it is very important that you answer the following questions as clearly and honestly as possible. All information will be completely confidential.

1. Are you currently smoking or have you within the last month smoked regularly (more than three times in one week) any type of cigarette?

2. Are you currently taking or have you within the last month taken any medication?

3. Are you currently using, or have you used within the last month an oral contraceptive?

4. Are you currently using or have you used any steroids or hormones within the last month?

5. Will you be participating in any intense physical activities (cross-country skiing, scuba-diving) on the weekend of January 17-20, 1986.?

APPENDIX D

Raw Data for all Subjects

Code #	Sex	Age	Ratio	D-Chol	D-Sat	%Fat	PA	PA rank
1	♀	18	2.76	422	.3	43	0	1
3	♀	18	2.93	211	.5	36	1092	3
4	♀	19	3.01	146	.7	28	0	1
6	♀	18	2.60	122	.5	24	0	1
7	♀	19	3.00	75	.7	30	480	2
8	♂	19	2.52	1061	.4	45	2210	3
9	♀	19	2.35	297	.2	43	1069	3
10	♀	20	3.39	106	.5	45	2040	3
11	♂	19	3.00	96	.7	36	0	1
13	♀	18	3.09	338	.3	34	2792	3
16	♂	21	3.47	297	.3	34	4600	3
21	♂	21	2.79	207	.2	34	2735	3
22	♀	18	2.54	230	.6	41	1450	3
23	♂	20	3.34	364	.1	38	2500	3
24	♀	19	3.39	203	.8	40	1400	3
25	♀	18	3.83	280	.5	28	250	2
26	♂	20	3.65	589	.3	41	2295	3
27	♂	18	4.19	2759	.5	44	2533	3
28	♀	21	3.28	226	.7	39	315	2
30	♂	23	3.50	391	.1	29	571	2
31	♀	18	1.93	211	.3	27	0	1
32	♀	22	3.26	347	.7	48	0	1
33	♂	22	2.86	840	.2	33	222	2
34	♀	19	2.13	180	.1	40	2250	3
36	♀	20	3.44	531	.2	40	0	1
39	♀	21	2.12	165	.5	43	0	1
40	♀	20	3.98	437	.2	45	720	2
42	♀	18	2.95	124	.3	27	3820	3
43	♂	21	2.66	415	.5	26	1934	3
44	♀	18	2.85	152	.5	28	0	1
47	♀	22	2.11	103	.3	39	426	2
49	♂	19	2.67	173	.2	37	0	1
50	♂	20	2.76	55	.5	38	3040	3
53	♀	18	4.14	189	.2	30	218	2